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SPOTLIGHT

Articles of Significant Interest Selected from This Issue by the Editors

Viral RNA Silencing Suppressors Bind Double-Stranded RNAs

RNA silencing (RNA interference) is an efficient antiviral system in plants. Therefore, successful infection requires expression of silencing suppressor proteins. Merai et al. (p. 5747–5756) show that many different plant virus silencing suppressors bind double-stranded RNAs (dsRNAs). This work suggests that these unrelated suppressors inhibit silencing similarly by sequestering dsRNAs, the key molecules of the silencing system. These findings also indicate that dsRNA binding is a general plant virus silencing suppression strategy, which has evolved many times independently.

Membranes Associated with the Severe Acute Respiratory Syndrome Coronavirus Replication Complex

RNA replication of positive-strand RNA viruses is commonly associated with (modified) intracellular membranes. Snijder et al. (p. 5927–5940) have analyzed the origin and ultrastructure of double-membrane vesicles that are induced upon severe acute respiratory syndrome (SARS) coronavirus infection and that have been implicated in viral RNA synthesis. Using a combination of immunofluorescence and immunoelectron microscopy, the presumed sites of viral RNA synthesis and virus assembly in infected cells were found to be distinct. The endoplasmic reticulum is the most likely membrane donor of the double-membrane structures that are thought to serve as the scaffold for the SARS-coronavirus replicative machinery.

Molecular and Functional Analysis of a Human Parvovirus B19 Infectious Clone

Parvovirus B19 (B19) is the only member of the *Parvoviridae* confirmed to cause disease in humans. Zhi et al. (p. 5941–5950) show that null mutants of the NS and VP1 proteins or deletion of the terminal hairpin sequence completely abolish B19 infectivity. Blocking expression of the 11-kDa protein significantly reduced B19 infectivity, and protein studies suggest that expression of the 11-kDa protein is critical for capsid production and trafficking in infected cells. These findings enhance our understanding of the key features of B19 replication and suggest new molecular targets for inhibiting B19 infection.

Cystine Noose of Respiratory Syncytial Virus Attachment Protein G Enhances Cytotoxicity

The respiratory syncytial virus (RSV) G protein was not considered necessary for the generation of RSV-specific cytotoxic T-lymphocyte (CTL) responses, because no anti-G CTL activity has been detected in mice and humans. Bukreyev et al. (p. 5854–5861) demonstrated that despite lacking $H\text{-}2^d$ restricted epitopes, G is critical for the generation of an effective CTL response against RSV. These findings reveal a novel function for RSV G, with potential implications for the generation of protective cellular responses and vaccine development.

Crystal Structures of Human Immunodeficiency Virus Type 1 (HIV-1) Neutralizing Antibody 2219 in Complex with Three Different V3 Peptides Reveal a New Binding Mode for HIV-1 Cross-Reactivity

Broadly neutralizing antibodies against human immunodeficiency virus type 1 (HIV-1) are rare, but their crystal structures have already revealed that each antibody utilizes a different strategy to recognize a wide variety of viral isolates. Stanfield et al. (p. 6093–6105) determined the crystal structure of human monoclonal antibody 2219, which recognizes the HIV-1 V3 loop. Structures of the Fab with three different V3 peptides show a novel mode of recognition whereby 2219 binds to a conserved face of the V3 β-hairpin but leaves the V3 crown sequence largely exposed. This study demonstrates yet another immune mechanism to achieve antibody cross-reactivity to viral isolates from different HIV-1 subtypes.

How a Morbillivirus Evades Innate Immunity and Inactivates the Adaptive Immune System

Experimental infections of ferrets with canine distemper virus (CDV) recapitulate many hallmarks of measles. To understand how a morbillivirus causes immunosuppression, von Messling et al. (p. 6084–6092) generated a CDV either unable to recognize its immune cell receptor or incapable of expressing either one, or both, candidate interferon antagonist proteins V and C. The immune cell receptor-blind CDV did not spread, suggesting that epithelial invasion is restricted to late stages of infection. The V-defective CDV was unable to inhibit cytokine responses and disseminate efficiently in lymphocytes, while C ablation had only minimal effects. Thus, V is the main CDV innate immunity evasion protein.